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The physiology of cardiac tamponade and implications for patient management

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ABSTRACT

Exceeding the limit of pericardial stretch, intrapericardial collections exert compression on the right heart and decrease preload. Compensatory mechanisms ensue to maintain hemodynamics in the face of a depressed stroke volume but are outstripped as disease progresses. When constrained within a pressurized pericardial space, the right and left ventricles exhibit differential filling mediated by changes in intrathoracic pressure. Invasive hemodynamics and echocardiographic findings inform on the physiologic effects. In this review, we describe tamponade physiology and implications for supportive care and effusion drainage.

1. Introduction

The normal pericardium prevents ventricular distension, promotes ventricular interdependence, and modulates hemodynamics and cardiac function [1,2]. In the late 19th century, cases of heart wounds leading to death not from exsanguination, but from compression due to small amounts of blood in the pericardial cavity were reported [1]. Tamponade occurs when intrapericardial collections compress cardiac chambers to sufficiently constrain filling and impact stroke volume (SV) and hemodynamics [3,4]. However, tamponade is not an "all-or-none" phenomenon and exists on a hemodynamic continuum mediated by several physiologic mechanisms and compensatory responses. With a moderate increase in pericardial pressure, the right atrium (RA) begins to collapse followed by early diastolic collapse of the right ventricle (RV) with a resultant reduction in cardiac output (CO). With disease progression, systemic arterial pressure decreases and cardiac arrest may ensue [5]. Here, we review the pathophysiologic mechanisms of cardiac tamponade and their implications for the clinical diagnosis and management of the patient.

2. Hemodynamic effects of pericardial compression

The pericardial pressure-volume relationship is a J-shaped curve (Fig. 1) [6]. Acutely, such as in cases of trauma or iatrogenic coronary perforation, the pericardial reserve volume is reached quickly thereafter small changes in volume cause large pressure variations. With slow accumulation of fluid, such as with malignancy, pericardial compliance increases and may accommodate over a liter of volume before operating on the steep portion of the curve [2]. Accordingly, the rate of fluid accumulation, pericardial compliance, and intrapericardial pressure, not volume alone, dictate the hemodynamic impact of an effusion.

Tamponade disproportionately impacts the thinner and more compliant right heart. As intrapericardial pressure rises, RV transmural pressure (intracardiac pressure - intrapericardial pressure) decreases and approaches zero (Fig. 2) [2,5]. The corresponding drop in preload results in a reduction of SV through a mechanism termed series ventricular interdependence. That is, the left ventricle (LV) can only eject what the RV delivers. While the measured right atrial pressure (RAP) will be elevated, it is not an accurate representation of RV preload as chamber distending pressures and volumes are low and venous return (VR) is restricted [5]. This reduction in right heart volumes, rather than direct LV compression, is the major contributor to the reduced cardiac output observed in tamponade [1].

Right atrioventricular interactions inform on several observations in tamponade [7]. The RA and RV exist within the pressurized and finite pericardial space [2,5]. In the early phases of tamponade, changes in total cardiac volume will impact total cavitary pressures (pericardial pressure + cardiac chamber pressure). A reduction in ventricular

Abbreviations: CO, cardiac output; LV, left ventricle; PPV, positive pressure ventilation; RA, right atrium; RAP, right atrial pressure; RV, right ventricle; SV, stroke volume; VR, venous return.

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volume during systole leads to a decrease in cavitary pressure promoting VR and RA filling in this phase of the cardiac cycle. During diastole, the increase in ventricular volume leads to a rise in cavitary pressure impeding right heart filling. In the late phase of tamponade, pericardial and diastolic chamber pressures are elevated and equalized. As pericardial pressures rise, VR progressively shifts to the systolic phase and becomes unimodal. The impaired emptying of the RA is observed as a blunted Y-descent in the central venous pressure tracing [3,7]. As pericardial pressures further increase, the Y-descent may become abolished.

Compensatory mechanisms of venoconstriction and salt and water retention serve to maintain VR in the face of elevated intrapericardial pressures. Blood volume expansion over time affords a degree of tolerance to pericardial compression. However, this comes at the expense of tissue edema. Adrenergic stimulation and resultant tachycardia promotes maintenance of CO in the setting of a limited SV [3]. As tamponade progresses, pericardial pressure approaches ventricular filling pressure eventually leading to elevation and equalization of chamber diastolic pressures with those of the pericardial sac [8]. The resultant decrease in VR is responsible for the reduction in cardiac output and hypotension. As compensatory mechanisms are outstripped, hemodynamic collapse may ensue.

3. Heart-lung interactions and ventricular interdependence in tamponade

Respirophasic cardiopulmonary interactions are responsible for several observed hemodynamic phenomena in tamponade physiology. Under normal circumstances, the decrease in intrathoracic pressure occurring with spontaneous inspiration increases the differential from the mean systemic filling pressure (MSFP) to the RA augmenting VR. As pleural pressure falls more than RAP, RA transmural pressure (RAP-pleural pressure) increases as does RV filling [9]. After several seconds, amplified RV SV is transmitted to the left heart through the process of series ventricular interdepenence [9]. Given this pulmonary transit time, the LV SV will be maximal during expiration.

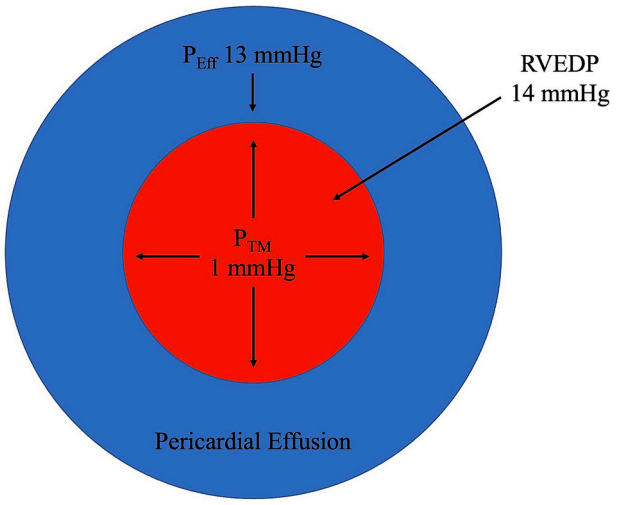

Fig. 2. Right ventricular transmural pressure in tamponade.
Diagram of the right ventricle (red circle) within a pericardial effusion. Under normal circumstances, the pericardial pressure is slightly negative and approximates the pleural pressure. In this case, the right ventricular end-diastolic pressure (RVEDP) measured with a pulmonary artery catheter is 14 mmHg (normal range 0–8 mmHg). However, as the pericardial effusion exerts a pressure (P_{Eff}) of 13 mmHg on the right ventricle, the right ventricular transmural pressure (P_{TM}), or RVEDP-PEff, is only 1 mmHg. Therefore, the effective distending pressure on the right ventricle is relatively low and right ventricular preload is reduced. Accordingly, patients with tamponade may have jugular venous distension despite an underfilled right heart because of the compressive effect of the effusion. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

The right and left ventricles interact not only in series, but in parallel through the shared interventricular septum. Under normal circumstances, as the pericardium is poorly expandable, the inspiratory increase in RV volume promotes leftward interventricular septal shift and

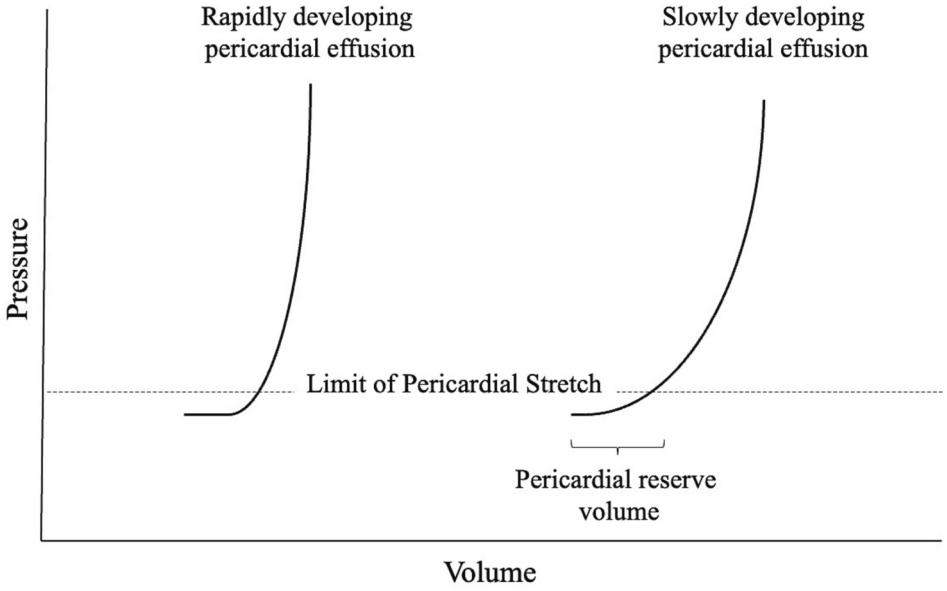

Fig. 1. Pericardial pressure-volume relationship.
The pericardial pressure-volume relationship is a J-shaped curve. As the pericardium is relatively inelastic, acute accumulation of fluid (as little as 150–200 mL in experimental models), rapidly exceeds the pericardial reserve volume (flat portion of the curve). Once the limit of pericardial stretch is reached, compliance is low and small changes in fluid volume produce significant increases in pericardial pressure (steep portion of the curve). With chronic effusions (right curve), the pericardial sac has time to stretch and accommodate a larger quantify of fluid (>1 L). Even with chronic effusions, the limit of pericardial stretch can be exceeded, and tamponade will occur. The pressure-volume relationship illustrates how the removal of a small quantity of fluid from a large effusion can relieve substantial pressure and normalize physiology.

therefore a slight decrease in LV SV during inspiration [10]. Simultaneously, there is decreased LV filling as blood pools in the pulmonary vasculature. In tamponade, outward RV free wall distension is further limited. Accordingly, the inspiratory increase in RV filling forces leftward interventricular septal bowing resulting in decreased LV filling, lower SV, and therefore a drop in systolic arterial pressure (Fig. 3). The 2 ventricles are 180° out of phase. As RV volumes rise, the LV is compressed and LV SV transiently falls [11]. Importantly, both parallel and series effects contribute to the inspiratory decrease in LV filling with inspiration. Pulsus paradoxus, a cornerstone in the diagnosis of tamponade, is an inspiratory decline in systolic blood pressure by >10 mmHg and is most readily appreciated with invasive blood pressure monitoring [10]. The paradox relates to Kussmaul's historic description of the radial pulse disappearing with inspiration despite the continued presence of an apical beat throughout the respiratory cycle. This is not a true paradox but an exaggeration of normal physiology [1]. Pulsus paradoxus may be observed in several conditions including asthma and hypovolemia [12]. Conversely, this finding may be absent with regional tamponade or in cases of elevated LV diastolic pressures precluding significant septal shift [1,10].

Under normal circumstances, positive pressure ventilation (PPV) is associated with a reduction in VR promoting rightward interventricular septal shift [13]. During a positive pressure breath, LV SV and therefore systemic arterial pressure will be maximal. With tamponade, this physiology is more pronounced given increased pericardial constraint and termed “reverse pulsus paradoxus.” [13] Positive pleural pressure is transmitted to the pericardial cavity. The use of positive-end-expiratory pressure (PEEP) leads to a further reduction in CO among patients with tamponade and should therefore be avoided in such scenarios [13].

4. Echocardiographic assessment of tamponade physiology

Physiology dictates echocardiographic findings observed with cardiac tamponade (Fig. 4). While a detailed discussion of this subject is beyond the scope of this review, select echocardiographic signatures merit attention [14-17].

- Diastolic RA collapse is an early sign of tamponade. The atrial walls are thinner and therefore more readily compressed than the ventricles. As pericardial pressure is higher during ventricular diastole than during systole, transient RA collapse will first appear during this phase of the cardiac cycle. When exceeding a third of the cardiac cycle, the duration of atrial inversion improved the specificity and positive predictive value of this finding for tamponade.
- Right ventricular early diastolic collapse, a specific finding, initially occurs during expiration when positive intrathoracic pressure decreases preload to the right heart. With disease progression, collapse extends to the inspiratory phase.
- Inferior vena cava plethora (defined as a diameter > 2.1 cm with <50% collapse over the respiratory cycle) reflects the inability of the pressurized RA to accept venous return and is highly sensitive for tamponade. False negative findings may be seen with low intravascular volume (“low pressure tamponade”).
- Mitral and tricuspid Doppler inflow variation are surrogates of pulsus paradoxus. With tamponade, rapid filling of the RV (represented by the *E*-wave measured at the tricuspid valve) is increased >40% during spontaneous inspiration compared to expiration. Simultaneously, inflow across the mitral valve decreases by >25% reflecting ventricular interdependence.

Absence of chamber collapse has a high negative predictive value for tamponade. However, elevated RV diastolic pressures such as in cases of pulmonary hypertension may resist collapse, conceal classic physiology, and protect from the compressive effects of pericardial collections. In such instances, atypical tamponade with left-sided chamber collapse may be evident [14,18]. Given heterogeneous studies and the imperfect predictive value of echocardiographic findings, integration of imaging findings with the clinical assessment is imperative.

5. Physiologic considerations in the management of tamponade

Early description of surgical pericardiotomy dates to the beginning of the 19th century. A few decades later, trocar insertion through an intercostal space to evacuate hemopericardium in a patient with a mediastinal neoplasm was described [1]. While the definitive treatment of tamponade is pericardial effusion drainage, temporizing and supportive measures may be required when the procedure is not immediately available.

Volume expansion may be expected to increase RV preload and therefore SV. In a clinical study of tamponade, volume expansion led to small increases in CO in 50% of patients while a third demonstrated reduction in CO with fluid administration. The benefit is largely limited to the provision of 250–500 cc fluid among those with relative hypotension (systolic blood pressure ⟨100) [19]. Accordingly, potentially with the exception of hypovolemia, volume expansion is rarely the solution in tamponade. Limited experimental and clinical studies inform on the effects of catecholamines in tamponade and the hemodynamic benefits are limited [20]. In cases of hemodynamic compromise, rapid

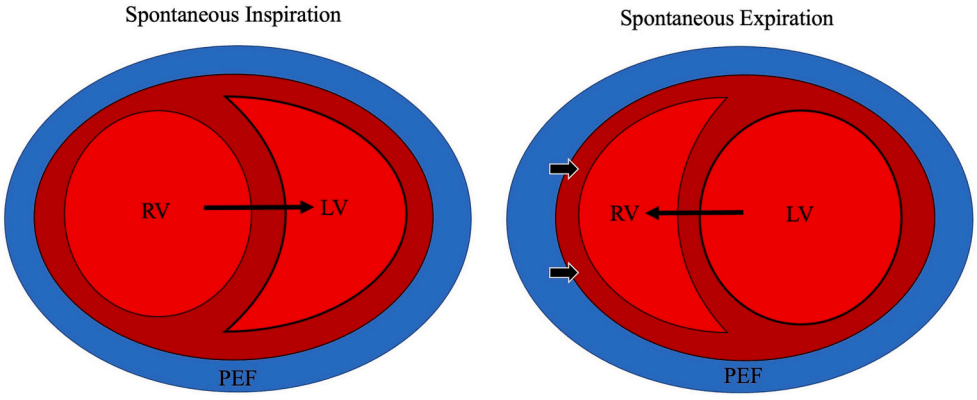

Fig. 3. Ventricular interdependence.
A. During spontaneous inspiration, right ventricular (RV) filling is increased. As the ventricles exist within a fixed pericardial space, RV filling occurs at the expense of the left ventricle (LV) causing interventricular septal shift toward the left (arrow) and restricting LV stroke volume. B. The opposite phenomenon occurs in expiration with causing the interventricular septum to shift toward the RV. Leftward arrowheads signify the compressive effects of the pericardial effusion on the RV. In tamponade, differential chamber filling over the respiratory cycle is responsible for a decline in systolic blood pressure > 10 mmHg with inspiration (pulsus paradoxus). PEF: pericardial effusion.

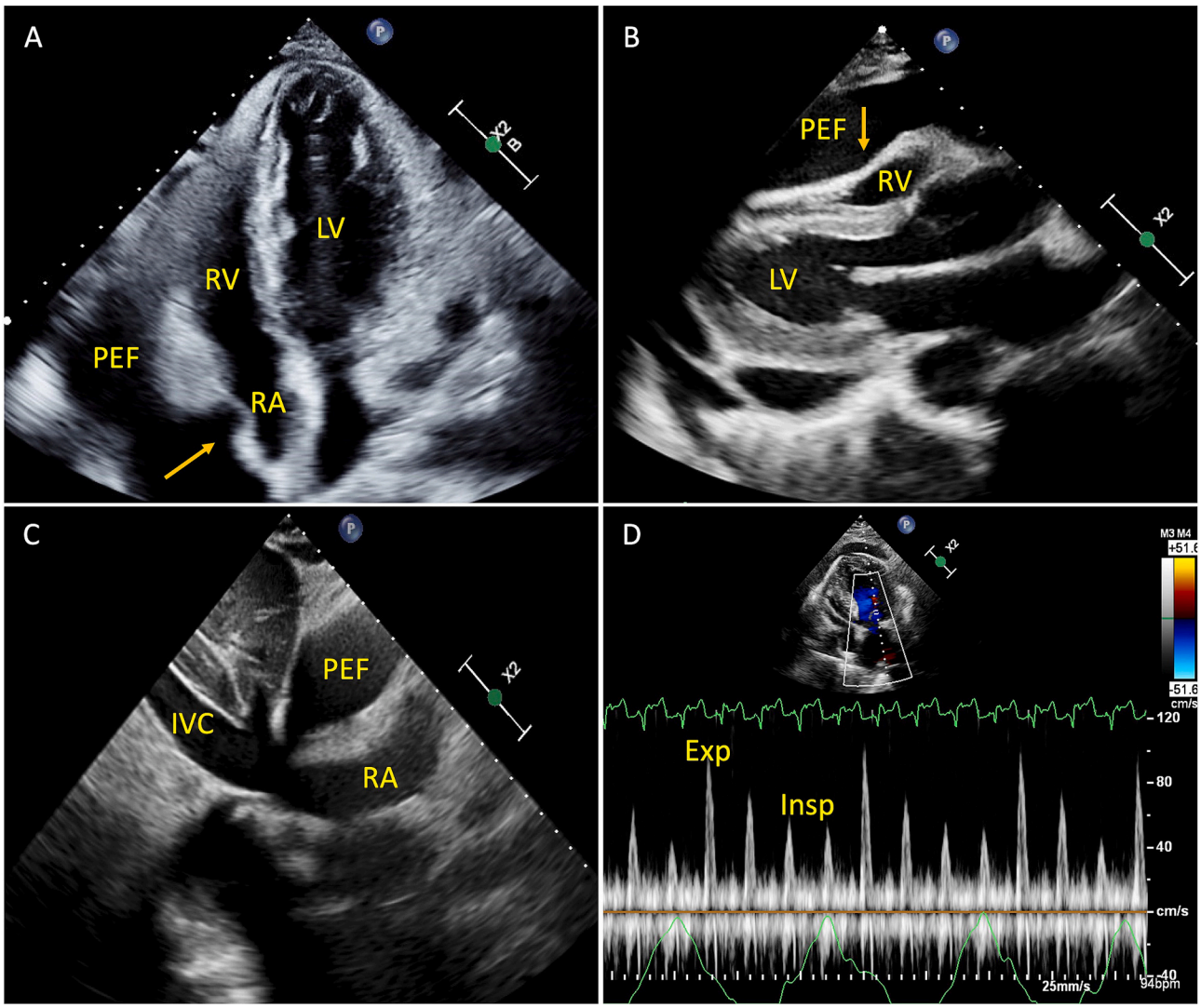

Fig. 4. Select echocardiographic signs in tamponade.
A. Apical 4-chamber view demonstrating right atrial (RA) systolic collapse, an early sign in tamponade (arrow – indented right atrial free wall). As disease progresses, collapse occurs over a greater duration of the cardiac cycle. B. Parasternal long-axis view depicting right ventricular (RV) diastolic collapse (arrow – indentation of the right ventricular free wall at the region of the outflow tract). This is the more specific echocardiographic finding in tamponade. While initially occurring during expiration, as disease progresses, collapse also involves the inspiratory phase. C. Subcostal view demonstrating inferior vena cava (IVC) plethora, a very sensitive sign in tamponade. Here, the IVC is dilated at 2.4 cm and does not collapse over the respiratory cycle. D. Pulse wave Doppler at the mitral valve illustrating respirophasic inflow variation. During spontaneous inspiration (Insp), increased filling of the RV occurs at the expense of left ventricular (LV) filling and diastolic flow across the mitral valve (*E*-wave) is decreased. The opposite occurs during expiration (Exp) as LV filling is enhanced. Here, inflow variation is approximately 50% (>25% consistent with tamponade physiology) and is a surrogate of pulsus paradoxus.
PEF: pericardial effusion.

relief of the effusion is most appropriate.

Increased intrathoracic pressure through PPV is transmitted to the pericardium, restricts venous return, further decreases RV transmural pressure, and may precipitate hemodynamic collapse. Loss of sympathetic tone with the induction of anesthesia may further lead to clinical deterioration. Accordingly, maintenance of spontaneous ventilation and effusion drainage prior to the institution of PPV is most appropriate in tamponade [13]. Maintaining compensatory adrenergic responses, avoiding bradycardia and myocardial depression are prime supportive measures.

With the limit of pericardial stretch reached, the steep pressure-volume relationship illustrates how the removal of even a small quantity of fluid from a large effusion may decompress the pericardial space and alleviate tamponade (Fig. 1) [1,21]. Expectedly, as the effusion is drained, systemic blood pressure and CO improve. Similarly, restoration of CO leads to a full recovery of the microcirculation and peripheral perfusion by contrast to models of septic shock where these abnormalities may persist following resuscitation [22]. Once pericardial pressure is below the RV end-diastolic pressure, further hemodynamic changes are no longer evident [1,8].

Very rarely, drainage of a chronic effusion may precipitate LV systolic dysfunction and result in shock, an entity termed pericardial decompression syndrome. While poorly understood, chronic impairment in filling may depress LV contractility; improved RV stroke volume following the relief of tamponade may contribute to the resultant clinical decline [23]. Right ventricular dysfunction has similarly been reported following pericardiocentesis yet is even less common. Unmasking of myocardial dysfunction with the alleviation of a high catecholamine state as well as the abrupt increase in RV preload are hypothesized mechanisms [24].

Pulmonary hypertension and elevated RV diastolic pressures may mask the classic findings in tamponade. Pericardial effusion drainage in

this context is controversial and is associated with periprocedural hemodynamic collapse. Plausibly, increased VR and resultant RV chamber expansion following the relief of tamponade may worsen LV cavity compression and lead to shock. In such cases, removal of small aliquots with pulmonary artery catheter guidance may be appropriate. Successful lowering of pericardial pressures just below diastolic chamber pressures without complete evacuation has been reported [18].

Drainage of the pericardial effusion is preferably performed percutaneously via catheter pericardiocentesis [25]. However, a surgical approach may be appropriate particularly in cases of suspected purulent pericarditis, penetrating trauma, or the need for pericardial biopsy. In cases of tamponade, a percutaneous approach may be performed more rapidly and obviate the need for general anesthesia and the associated risk required for surgical drainage. Pericardiocentesis should be guided by echocardiography to identify the area of maximal fluid collection and to determine needle trajectory. The instillation of agitated saline can confirm catheter position within the pericardial space sonographically. Alternatively, a fluoroscopic technique provides for visualization of needle entry in relation to the pericardium, and allows for the injection of small aliquots of contrast to confirm location [25]. A guidewire may be advanced through the needle and confirmed to be within the pericardial space with this imaging modality prior to catheter insertion.

Pericardial tamponade post cardiac surgery may manifest distinctly from classic tamponade. May features of tamponade depend on the presence of a circumferential pericardial effusion and equal transfer of pressures to all chambers [26]. Intrapericardial hematomas may cause regional compression of the right or left atria. Further, such intrapericardial collections may be missed with transthoracic echocardiography. Accordingly, a high index of suspicion for tamponade in this patient population is necessary. Alternative imaging modalities such as transesophageal echocardiography or CT may be required [27]. In select circumstances, the patient may need to be brought back to the operating room for re-exploration.

6. Conclusions

Pericardial effusions precipitate hemodynamic changes via compression and restriction of chamber filling. Characteristic physiology observed with cardiac tamponade is mediated by increased pericardial constraint and heart-lung interactions leading to differential filling of the ventricles. Tamponade exists on a spectrum best appreciated through the combination of echocardiographic findings and clinical manifestations. Physiology dictates patient response to supportive measures and informs on the effects of pericardial effusion drainage.

Role of the funding source

None.

CRediT authorship contribution statement

Eugene Yuriditsky: Conceptualization, Supervision, Writing – original draft, Writing – review & editing. **James M. Horowitz:** Conceptualization, Supervision.

Declaration of Competing Interest

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